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Animal Health Hazards of Concern During Natural Disasters

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Contributors

USDA: Animal and Plant Health Inspection Service (APHIS), Veterinary Services (VS), Centers for Epidemiology and Animal Health (CEAH), Center for Emerging Issues (CEI)

Reginald Johnson, 970-490-7896, reginald.a.johnson@aphis.usda.gov

Miloslav Muller, 505-761-3160, miloslav.muller@aphis.usda.gov

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Colleen S. Bruning-Fann, Area Epidemiology Officer, USDA/APHIS Veterinary Services, Lansing, MI

David M.Ewey, Veterinary Medical Officer, USDA/APHIS Veterinary Services, Sacramento, CA

Dana Nelson, Area Epidemiology Officer, USDA/APHIS Veterinary Services, Sacramento, CA

Whitney A. Ross-Mauer, Epidemiologist, Kent County Health Department, Grand Rapids, MI

Gary E. Stevens, Area Epidemiology Officer, USDA/APHIS Veterinary Services, Lincoln, NE

Carol A. Tuszynski, Agricultural Economist, USDA/APHIS Veterinary Services, Fort Collins, CO

Copies

For copies of this publication, please contact the Centers for Epidemiology and Animal Health:

Telephone	(970) 490-8000
FAX	(970) 490-8099
Internet	www.aphis.usda.gov/vs/ceah/cei

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List of Abbreviations

EEE, eastern equine encephalomyelitis
FEMA, Federal Emergency Management Agency
SLE, St. Louis encephalitis
VEE,Venezuelan equine encephalomyelitis
WEE, western equine encephalomyelitis

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Executive Summary

Damage from seemingly more frequent and more severe weather calamities and other natural phenomena during the decade from 1990 to 1999 lead to 460 Presidential disaster declarations. The number of disaster declarations that were issued for 1990 to 1999 was approximately double the number that was issued for 1980 to 1989 and for all preceding decades on record. An important concern during natural disasters is the potential for outbreaks of diseases in animals and humans. The animal diseases for which there is concern may be classified broadly into two categories, infectious hazards and non-infectious hazards. Examples of infectious hazards commonly discussed in the aftermath of hurricanes or other disasters resulting in flooding include the mosquito-borne diseases (e.g., eastern equine encephalomyelitis), leptospirosis, anthrax, botulism, cryptosporidiosis, giardiasis and hoof rot. Frequently discussed non-infectious hazards of animals during natural disasters include traumatic injuries, aspiration pneumonia, and toxic and sewage-related gastroenteritis. There is little rigorous scientific documentation that the incidence of animal diseases increases substantially, either during or shortly after natural disasters. The absence of such scientific documentation suggests that there could be a significant disparity between the perceptions and the realities of the incidence of animal diseases during natural disasters. Thus, a valuable service that health professionals can provide during natural disasters is communication to emergency management agencies, the news media and the public of accurate scientific information about the potential risk of infectious and non-infectious hazards to animal and human health.

The objective of this paper is to describe some of the major natural disasters that have occurred in the U.S. during recent years and to review some infectious and non-infectious hazards that, at the very least, are perceived to be related directly to natural disasters. The number and types of natural disasters, the basic ecology and epidemiology of several infectious hazards that are thought to be affected by the climatic and environmental changes during natural disasters, and the impact of natural disasters on some non-infectious hazards of animals are presented.

The U.S. experienced 186 Federally-declared, natural disasters during years 1998-2000. Thunderstorms and floods comprised slightly more than 50 percent of the total number of these natural disasters. Regarding infectious hazards, during the past 25 years there was only one natural disaster involving flooding (i.e., the Red River flood of 1975) to which significant animal morbidity and mortality due to arbovirus disease was attributed. There is minimal to no scientific evidence that links natural disasters in the U.S. to significantly consistent increases in the incidence of other infectious diseases of animals such as anthrax, leptospirosis, and cryptosporidiosis. While not documented, the destruction caused by some natural disasters might in fact destroy ecosystems that normally harbor pathogens and their vectors and consequently decrease, not increase, the risk of outbreaks of infectious disease. Regarding non-infectious hazards, sporadic incidents of morbidity and mortality due to drowning, heat waves, and traumatic injuries have been documented more clearly. Thus, the potential economic impact of non-infectious hazards arising from natural disasters may be greater than the economic impact of infectious hazards. While vigilant surveillance for outbreaks of infectious hazards during natural disasters should not be discouraged, there should be appropriate resources allocated towards the resolution of issues related to the prevention, control and treatment of non-infectious hazards.

Introduction

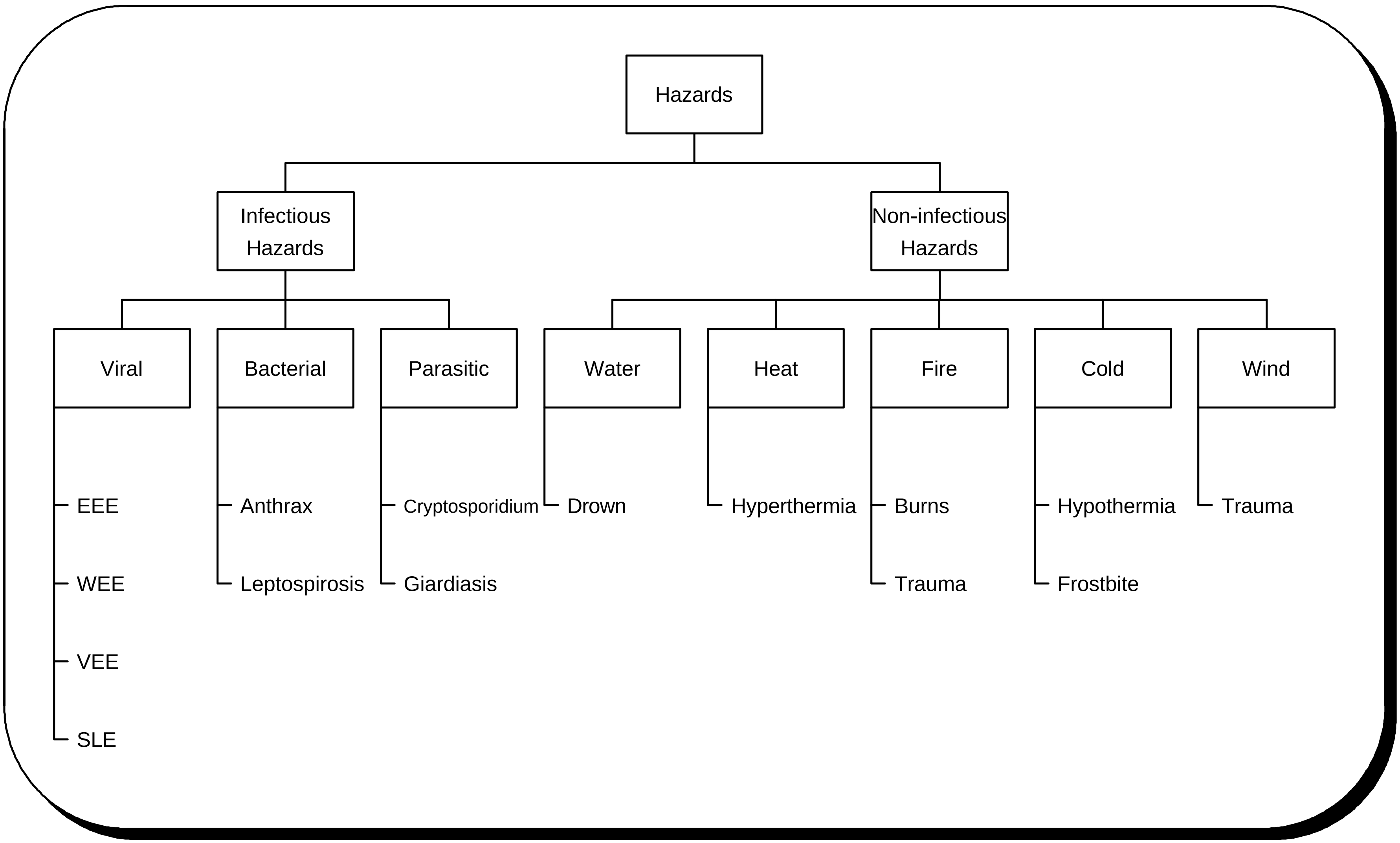
Damage from seemingly more frequent and more severe weather calamities and other natural phenomena during the decade from 1990 to 1999 lead to 460 Presidential disaster declarations (Federal Emergency Management Agency[FEMA], 2000a). The number of disaster declarations that were issued for 1990 to 1999 was approximately double the number that was issued for 1980 to 1989 and all preceding decades on record. For the 1990-1999 period, the FEMA spent more than $25.4 billion for declared disasters and emergencies, compared to $3.9 billion (current dollars) in disaster aid for the 1980-1989 period. Hurricanes and typhoons were the most costly of the weather-related events during the 1990s. A total of 88 declarations were issued for these storms, including a single-year record of 19 declarations in 1999. The flooding that resulted from severe storms and other causes of flooding was the most frequently declared type of disaster. Approximately $7.3 billion in funding was committed by FEMA to response and recovery from flooding during 1990 to 1999.

Natural disasters can impact livestock and companion animals. Pigs, chickens and turkeys were killed in the wake of Hurricane Floyd in North Carolina in 1999. A disaster relief management team was established by the North Carolina State University College of Veterianry Medicine for companion animals including dogs, cats and horses during Hurricane Floyd. A similar team provided care for horses during Hurricane Andrew in Florida in 1992. These events have highlighted the need to include livestock and companion animals in disaster management plans. The importance of caring for animals during disasters has bearing on two significant issues, public health and animal well-being (Heath, 1999). The close relationship between companion animals and their owners as well as livestock and their owners (Monti, 2000) suggests that providing care for animals in disasters should be an integral component of providing care for humans.

An important concern during natural disasters is the potential for outbreaks of diseases in animals and humans. The animal diseases for which there is concern may be classified broadly into two categories, infectious diseases and non-infectious diseases **(Figure 1)**. Examples of infectious diseases commonly discussed in the aftermath of hurricanes or other disasters resulting in flooding include the mosquito-borne diseases (e.g., eastern equine encephalomyelitis), leptospirosis, anthrax, botulism, cryptosporidiosis, giardiasis and hoof rot (Nasci and Moore, 1998; Heath, 1999; University of North Carolina, 1999). Frequently discussed non-infectious diseases of animals during natural disasters include traumatic injuries, aspiration pneumonia, and toxic and sewage-related gastroenteritis.

The potential for animal-related disease outbreaks during natural disasters is discussed in media reports frequently (Heath, 1999). However, there is little confirmed documentation ("scientific" or otherwise) that the incidence of diseases increases substantially, either during or shortly after natural disasters. The absence of such scientific documentation suggests that there could be a significant disparity between the perceptions and the realities of the incidence of animal diseases during natural disasters. Thus, a valuable service that health professionals can provide during natural disasters is communication to the news media and the public of accurate scientific information about the potential risk of infectious and non-infectious hazards to animal and human

Figure 1. Animal-health hazards of concern and their consequences during natural disasters.



health. Being equipped with accurate information about the potential risk of disease will permit emergency management agencies to direct their energy and resources towards issues that truly are of utmost importance during disasters.

Objective

The objective of this paper is to describe some of the major natural disasters that have occurred in the U.S. during recent years and to review some infectious and non-infectious hazards that are perceived to be related directly to these natural disasters. Specifically, the number and types of natural disasters, the basic ecology and epidemiology of several infectious hazards that are thought to be affected by the climatic and environmental changes during natural disasters, and the impact of natural disasters on some non-infectious hazards of animals will be presented.

Target Audiences

The project was undertaken with two audiences in mind. In the interest of generality, brevity and handiness, the contents of this paper were written for veterinary health professionals as well as other professionals who are involved, or who expect to be involved, in addressing animal health issues that may arise during natural disasters. The section "Infectious Hazards of Concern During Natural Disasters" is comprised of excerpts from a series of seven supplemental readings (readings in preparation) in which the ecology and epidemiology of each infectious hazard is discussed in greater detail. Beyond the scope of this paper, the supplemental readings may be beneficial to those readers who, for purposes other than natural disasters, have an interest in the basic ecology and epidemiology of the infectious diseases that are discussed herein. Although humans are susceptible to each and every disease that has been included in this paper, the primary focus of the paper is disease in domestic species, and to a lesser extent, wildlife species. For comparative purposes, there will be an occasional reference to similar investigations in human populations. Extensive bodies of literature about these diseases in humans are available elsewhere.

Expected Outcome

The expected outcome for the reader is a greater awareness of : **(1)** the complexity of transmission of infectious hazards that typically are of concern during natural disasters, **(2)** the multitude of environmental and biologic factors that may be involved in the transition of these infectious hazards from a sporadic to an enzootic state, and from an enzootic to an epizootic state, and **(3)** the relative potential impact of infectious hazards versus non-infectious hazards during natural disasters.

Summary of Federally-declared Natural Disasters In The U.S., 1998-2000

According to the United States Federal Emergency Management Agency, there are 17 forms of disasters, 13 of which are broadly categorized as natural disasters and 4 of which are categorized as technological disasters **(Table 1)**. The U.S. experienced 186 Federally-declared, natural disasters during years 1998-2000. There were 58 Federally-declared natural disasters during year 1998, 61 during year 1999, and 67 during year 2000 **(Figure 2)**. The most prevalent Federally-declared natural disaster was the thunderstorm. Thunderstorms and floods comprised slightly more than 50 percent of the total number of these natural disasters **(Figure 3)**. Wildfires, mudslides, and earthquakes were among the least prevalent of these natural disasters during the three-year period. After being stratified by year, the distribution of these natural disasters was similar to the non-stratified distribution (i.e., from one year to the next, thunderstorms and floods remained as the most prevalent types of natural disasters) **(Figure 4)**.

Numerous states may be affected by a Federally-declared natural disaster such as a hurricane. Because the geographic unit of concern that is used by FEMA is the state (or its equivalent), a single natural disaster such as a hurricane may be recorded several times, but by a different state each time. Thus, Hurricane Floyd was recorded as at least eight different natural disaster declarations during year 1999.

Table 1. Types of natural and technological disasters, according to the United States Federal Emergency Management Agency (FEMA).

Type of Disaster	Form
Natural	Earthquakes
	Extreme heat
	Fire safety (during or after a disaster)
	Floods and flash floods
	Hurricanes
	Landslides and mudflows
	Tornadoes
	Tsunamis
	Volcanoes
	Wildland fires
	Winter driving
	Winter storms
	Thunderstorms and lightning
Technological	Hazardous materials
	House fires; building fires
	Nuclear power plant emergency
	Terrorism

Source. http://www.fema.gov/ptc/prep.htm. . 2001

Figure 2. Annual number of Federally-declared, natural disasters in the U.S. during years 1998-2000 (Source http://www.gismaps.fema.gov/).

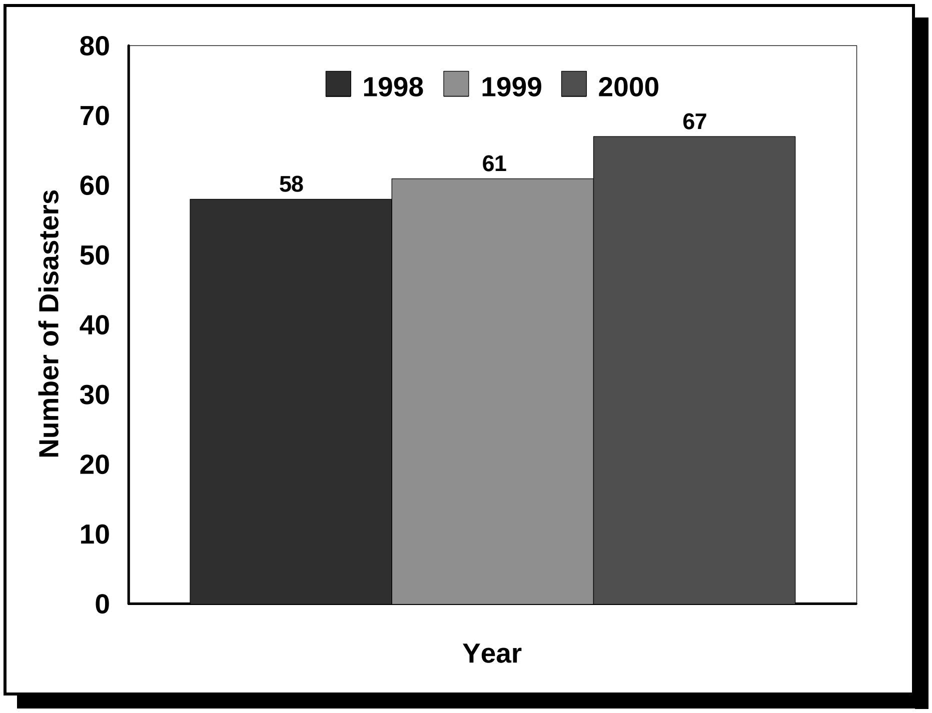


Figure 3. Types of Federally-declared, natural disasters in the U.S. during years 1998-2000 (Source http://www.gismaps.fema.gov/).

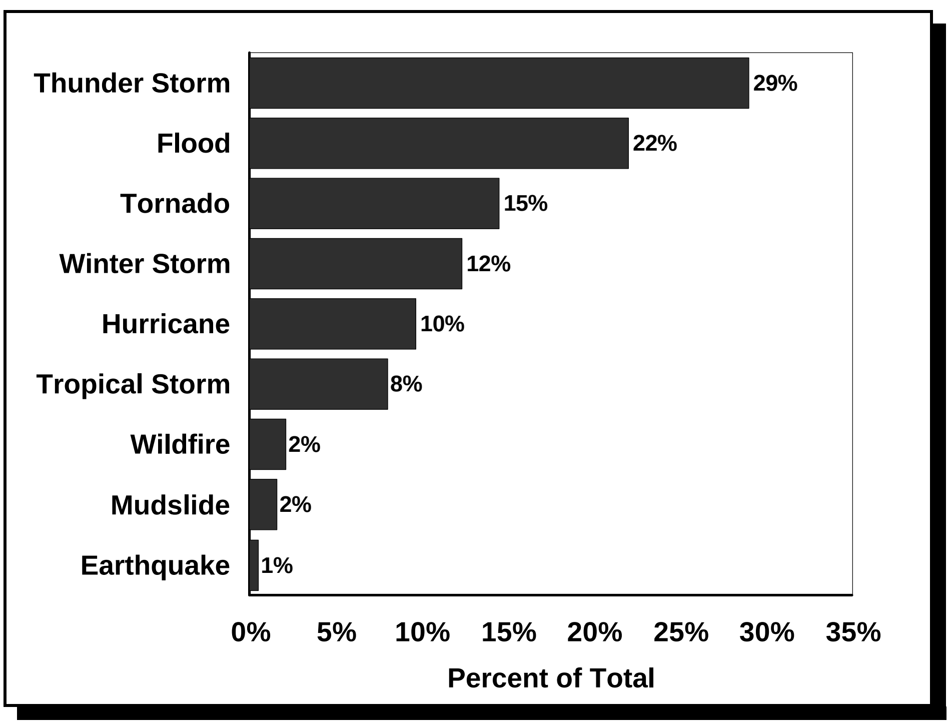
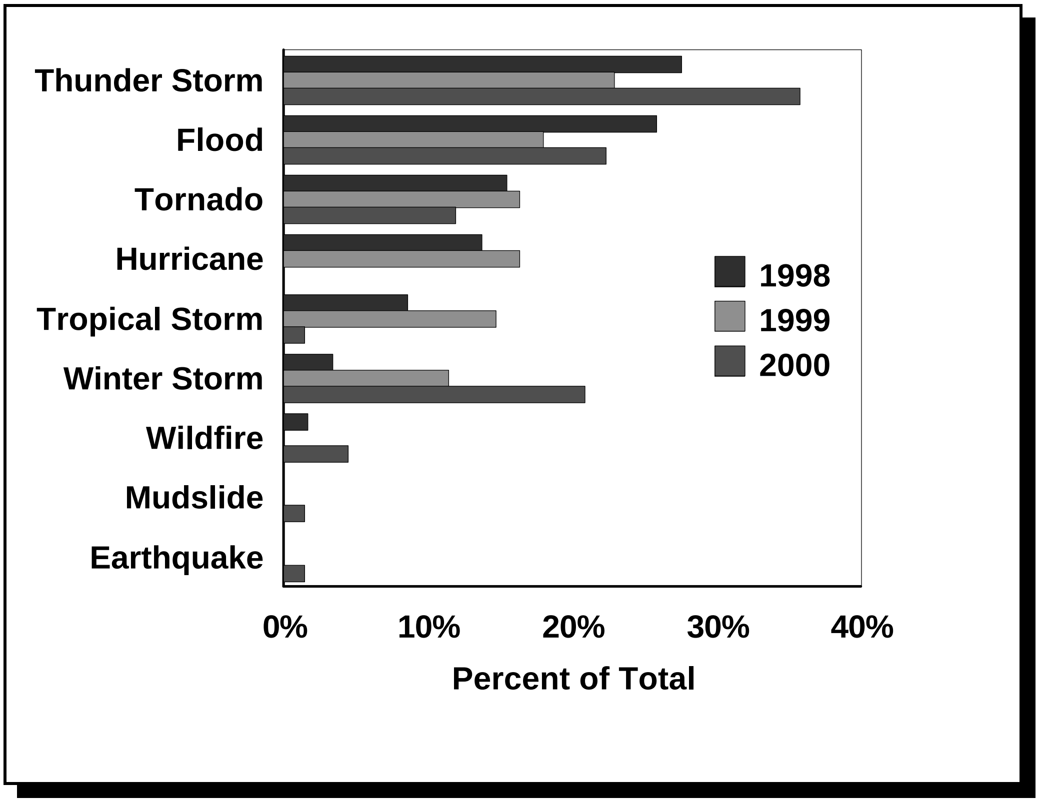


Figure 4. Types of Federally-declared, natural disasters in the U.S. by year during years 1998-2000 (Source: http://www.gismaps.fema.gov).



Infectious Hazards of Concern During Natural Disasters

One possible outcome of natural disasters is alteration of ecosystems that harbor pathogens for infectious diseases of animals, as well as vectors of these pathogens. One of the most common forms of natural disasters is the flooding that results from excessive rainfall. Examples of infectious diseases of animals that become of concern due to proliferation of mosquitoes during and after flooding are the arbovirus diseases such as eastern equine encephalomyelitis and western equine encephalomyelitis, (Nasci and Moore, 1998; Heath, 1999) **(Figure 1)**. Bacterial and parasitic diseases of animals that are commonly discussed in the aftermath of floods, hurricanes, droughts and similar natural disasters are leptospirosis, anthrax, botulism, cryptosporidiosis, giardiasis and hoof rot (Nasci and Moore, 1998; Heath, 1999; University of North Carolina, 1999). Various ecological, geographical, and climatic factors, some of which have been studied more intensely than others, have been observed during outbreaks of these diseases. Each of these groups of factors may be influenced to varying degrees by natural disasters.

During natural disasters, animal health professionals are expected to provide a range of veterinary services to protect animal and human health. One of these services includes prevention and control of epizootic, zoonotic and food-borne diseases of animals and humans (Bartels and Thornton, 1987; Shomer, 1987; Huxsoll et al., 1987; Heath 1999). To achieve their goals, at least a fundamental understanding of the ecology and epidemiology of potentially infectious hazards is indispensable. This information can be used to assess the potential influence of natural disasters on these infectious hazards and is provided below.

Arbovirus Diseases

The word “arbovirus” is used to describe a diverse array of viruses that share a common characteristic, namely transmission by arthropod vectors (Monath, 1988). The arbovirus group includes at least 500 viruses, most of which have been assigned to five families, Togaviridae, Flaviviridae, Bunyaviridae, Reoviridae, and Rhabdoviridae. More than 100 arboviruses have been associated with naturally-acquired diseases in domestic animals and humans. Some viruses of importance to animal and human health in the western hemisphere are listed in **Table 2**.

Ecology of Eastern Equine Encephalomyelitis

Eastern equine encephalitis (EEE) is caused by viruses that can be transmitted to equines and humans by the bite of an infected mosquito. EEE viruses are alphaviruses that were first identified in the 1930's. In addition to horses and humans, EEE viruses can produce severe disease in some birds such as pheasant, quail, the ostrich and the emu (Centers for Disease Control and Prevention, 1999).

Table 2. Serogroup, taxonomy, virus, primary vector, geographic distribution, and diseases of zoonotic and non-zoonotic arboviruses that are indigenous to Canada, the United States, Central America, or South America.

Serogroup	**Family**	**Genus**	**Virus**	**Vector**	**Geographic Distribution**	**Disease**
Group A	Togaviridae	Alphavirus	Eastern Equine Encephalitis (EEE)	Mosquitoes	Canada, U.S.	Encephalitis, fever
			Everglades (VEE II)	Mosquitoes	Florida	Encephalitis, fever
			Venzuelean Equine Encephalitis (VEE)	Mosquitoes	South America, Central America	Encephalitis, fever
			Western Equine Encephalitis (WEE)	Mosquitoes	Canada, U.S.	Encephalitis, fever
Group B	Flaviviridae	Flavivirus	Powassan (POW)	Ticks	Canada, U.S.	Encephalitis
			St. Louis encephalitis (SLE)	Mosquitoes	Canada, U.S.	Encephalitis, fever
Group CAL	Bunyaviridae	Bunyavirus	California encephalitis	Mosquitoes	Canada, U.S.	Encephalitis, fever
			Jamestown Canyon	Mosquitoes	Canada, U.S.	Encephalitis, fever
			LaCrosse (LAC)	Mosquitoes	U.S.	Encephalitis, fever
			Snowshoe hare	Mosquitoes	Canada, U.S.	Encephalitis, fever
CTF	Reoviridae	Coltivirus	Colordo Tick Fever (CTF)	Ticks	Canada, U.S.	Fever, myalgia

Source: From Calisher, *Clinical Microbiology Reviews*, 1994. Revised, with permission.

Regarding insect and arthropod vectors, EEE viruses have been isolated from mosquitoes, gnats, horse flies, blackflies, mites, and lice. However, the majority of EEE viruses that have been isolated were from 27 different species of mosquitoes, and 80 percent of the isolates were from a single species, *Culiseta melanura* (Morris, 1994). The primary habitats for EEE viruses are lowlands (Morris, 1994). Endemic EEE swamps located in Florida, Maryland, New Jersey, New York, and Michigan are characterized by muck-peat soils that are dominated by hardwoods. These hardwoods have a preference for wet, mucky habitats, and they provide a root system that supports oviposition and larval development by *C. melanura*. *C. melanura* larvae require a source of water that is darkly shaded and that contains a high concentration of organic matter. However, *C. melanura* also breeds in un-shaded waters of cypress swamps in Florida during the rainy season. The mosquito larvae that overwinter in these habitats develop into pupae. Adult mosquitoes emerge from the pupae, first in February and March in Florida, and in April through May in the northern U.S. Female adult mosquitoes may become dispersed as far as five miles from the swamp from which they originated.

Specific weather conditions that precipitate disease outbreaks attributable to EEE viruses have not been established clearly. However, outbreaks usually are observed during periods of hot, rainy weather; these conditions are ideal for expansion of *C. melanura* and other mosquito populations (Walton, 1992; Nasci et al., 1993). Outbreaks of EEE are expected to occur from midsummer to late summer, with August being the peak month of incident cases. An epidemiological investigation of EEE in Michigan showed that the incident equine cases were most frequent during September (Ross and Kaneene, 1996). Although outbreaks occur during the summer, the environmental temperature alone has **not** been shown to be a valuable predictor for EEE outbreaks. The annual mean environmental temperatures in Michigan were approximately normal during each of the years prior to and each of the years of an outbreak of EEE (Ross and Kaneene, 1996).

An annual excess in precipitation of 20-plus centimeters of rainfall for two consecutive years, (1) the year preceding the outbreak and (2) the year of the outbreak, has been reported as a predictor of EEE outbreaks, but the source of this data was limited to a few states in the U.S. (Grady et al 1978). Others have attempted to create a predictive model for EEE using rainfall (Letson et al. 1993). The predictive value of the models by Letson et al. never exceeded 50 percent, suggesting that they would not have been very useful in making the predictions about when outbreaks of these diseases would occur relative to the volume of rainfall. The annual precipitation in Michigan was rarely 10 centimeters (approximately 8 percent) greater than expected in each of the years prior to and each of the years of an outbreak of EEE. This eight percent increase does represent above-average annual precipitation, but there is no indication that it lead to flooding that was equivalent to that of a natural disaster. An analysis of precipitation data from Michigan showed that precipitation in Michigan also was increased during late summer and early fall of the year preceding the outbreak, and it was increased during the summer of the outbreak (Ross and Kaneene, 1996). In Massachusetts and New Jersey, there were outbreaks of EEE when there was unusually heavy rainfall during late summer and early fall of the year preceding the outbreak, and during the summer of the year of the outbreak (Grady et al., 1978).

Although there have not been consistent observations of EEE outbreaks during specific weather conditions, a specific type of land cover is one factor that has been observed consistently in the habitats occupied by EEE virus vectors and wild-bird reservoir hosts for the virus (Morris et al., 1980; Emord et al., 1984). The hydrographic (versus geographic) regions that are classified as "incomplete drainage-lake type" regions are regions in which the glaciated surface does not permit the streams to drain completely. The regions are characterized by numerous small lakes, swamps, and bogs that are connected by streams and that usually have dry land between the lakes, swamps, and bogs. Outbreaks of EEE have been observed in these hydrographic regions in New York, Ohio and Michigan (Morris et al., 1980; Nasci et al., 1993; Ross and Kaneene, 1996).

For more information about the epidemiology and ecology of EEE, you are referred to the supplemental readings listed in **Appendix 1**.

Ecology of Western Equine Encephalomyelitis

The alphavirus of western equine encephalitis (WEE) was first isolated in California in 1930 from the brain of a horse with encephalitis (Hardy, 1987). WEE remains an important cause of encephalitis in horses and humans in North America, mainly in western parts of the USA and Canada. WEE viruses also have been isolated from a variety of mammal species (Centers for Disease Control and Prevention, 1999).

Epizootics of WEE have been reported mostly in the states west of the Mississippi River in the U.S., and in the provinces of Alberta, Manitoba, and Saskatchewan in Canada. However, both the ecology and epidemiology of WEE have been studied most intensively in the Central Valley of California (Hardy, 1987). Two mosquito species are involved in the summer transmission cycle of WEE viruses in California, *Culex tarsalis* and *Aedes melanimon*. These two species of mosquitoes prefer to breed in sunlit grassy marshes and in open pools of stream beds, especially in irrigated areas where seepage and improper flooding of agricultural lands create favorable breeding sites (Walton, 1992).

Although flooding due to a natural disaster such as excessive rainfall may favor transmission of WEE viruses, "human-made disasters" due to construction of irrigation systems for cropland may be just as important, if not more important, in the transmission of WEE viruses. The impact of these irrigation districts on WEE virus transmission has been demonstrated rather clearly in the Central Valley of California (Reeves and Milby, 1989). Approximately 51 percent of *Culex tarsalis* larval collections during a two-year plus period were from agricultural irrigation water sources including waste water and residual water, pools of water due to leakage and seepage from the irrigation canals, irrigation canals and ditches themselves, standpipes and valve housings, and reservoirs. Incident cases of WEE and SLE in humans in California were greatest during this two-year period. The remaining sources of water from which *C. tarsalis* larvae were collected during this epidemic period were miscellaneous sources of agricultural water, domestic water, industrial water, municipal water, natural streams, and recreational waters (e.g., duck hunting clubs). More than 90 percent of mosquito breeding sites that were located in irrigated, agriculturally developed, and partially urbanized areas were sites that were constructed by humans. *C. tarsalis* populations will flourish even in deserts, if the deserts are reclaimed and irrigated. The altered desert environment not only supports a vector population such as *C. tarsalis,* but the environment also provides the vegetative cover and food supply to support birds that are essential hosts for WEE viruses.

A qualitative model was developed by Gordon-Smith to explain the factors that influence the **enzootic** (versus epidemic) transmission of WEE viruses between its vertebrate maintenance hosts and from these maintenance hosts to humans (Gordon-Smith, 1987). The model was derived from comprehensive studies of WEE virus transmission in numerous field and laboratory studies in Kern County California during an approximate 45-year period between 1945 and 1985. One example of the factors included in the model, factors that may be influenced by environmental conditions, is the relationship between the ambient temperature and survival of mosquitoes. The longevity of mosquitoes is influenced by both ambient temperature and relative humidity, specifically the relative humidity of the vector's resting environment and the atmospheric relative humidity during its feeding periods. The favorable range of relative humidity during the season of WEE virus transmission in Bakersfield, California is narrow, roughly 35 to 55 percent; therefore, it is logical that *Culex tarsalis* prefers to reside in humid refuges such as culverts. The relative humidity of the vector's resting environment can be correlated with the atmospheric relative humidities of each season of the year. Thus, the survival of *C. tarsalis* will be greater during August and September, versus June and July. Numerous other factors, **perhaps 50 or more**, may influence the ecology and epidemiology of WEE (Reeves, 1967).

For more information about the epidemiology and ecology of WEE, you are referred to the supplemental readings listed in **Appendix 1**.

Ecology of Venezuelan Equine Encephalomyelitis

Like EEE and WEE viruses, Venezuelan equine encephalitis (VEE) virus is an alphavirus that causes encephalitis in horses and humans. VEE is an important veterinary and public health problem that is confined primarily to Central and South America. Occasionally, large regional epizootics and epidemics can occur, resulting in thousands of equine and human infections. A large epizootic that began in South America in 1969 spread to Texas in 1971. At least 200,000 horses died during that outbreak (Walton and Grayson, 1989; Centers for Disease Control and Prevention, 1999).

Culex mosquitoes of the subgenus Melanoconion, the primary **enzootic** vector of VEE viruses, reside in tropical and subtropical swamps and forests throughout North and South America. These mosquitoes breed in pools of water or meandering streams that are closely associated with aquatic plants such as water lettuce (Johnson and Martin, 1974). Some adult mosquitoes remain within naturally shaded, moist locations while others may invade human residences. Rodents are the mosquito's primary vertebrate partner in maintaining VEE viruses in the swamp and forest habitats.

Mosquitoes of several different genera including *Mansonia*, *Psorophora*, and *Aedes* are involved in **epizootic** transmission of VEE (Walton and Grayson, 1989). The favored ecological zones for epizootics of VEE are tropical dry forest and tropical thorn forest, both of which also support agricultural and cattle ranching enterprises. VEE epizootics occur typically during the rainy season in these ecological zones. In addition to rainfall, a relative humidity of 90 percent and an ambient temperature of 26.6º C are other environmental factors that may influence VEE epizootics. Frequently, VEE epizootics are observed during periods of unusually intense rainfall (Trapido, 1972; Johnson and Martin, 1974; Walton, 1981), but these levels of excessive rainfall would not necessarily be the equivalent of the excessive rainfall that could be experienced during a natural disaster.

For more information about the epidemiology and ecology of VEE, you are referred to the supplemental readings listed in **Appendix 1**.

Ecology of St. Louis Encephalitis

St. Louis encephalitis (SLE) virus, a Group B arbovirus, is a member of the family Flaviviridae and the genus Flavivirus. Unlike EEE, WEE, and VEE, SLE is a rarely diagnosed disease of animals, but because chickens and wild birds are susceptible to SLE virus infection, they are used as sentinels to predict transmission of SLE virus in humans. Serological evidence of natural transmission of SLE virus was detected in an emu flock that was suspected of experiencing morbidity due to an arbovirus (Day and Stark, 1996; Day and Stark, 1998) and in small mammals (Day et al., 1996). In humans, SLE is the leading cause of epidemic flaviviral encephalitis in the United States, and it is the most common mosquito-transmitted human pathogen in the U.S. While periodic SLE epidemics have occurred in the U.S. in the Midwest and southeast only, SLE virus is distributed throughout the lower 48 states (Centers for Disease Control and Prevention, 1999). Some aspects of the ecology and epidemiology of SLE in humans may be extremely beneficial in thoroughly understanding naturally-acquired SLE virus infections of animals that may occur in the future, as well as arbovirus infections of animals such as WEE, EEE and VEE.

Culex pipiens and *Culex quinquefasciatus* are the primary vectors of SLE virus in the Ohio-Mississippi basin and eastern Texas. *C. pipiens* and *C. quinquefasciatus* breed in polluted waters, especially where sanitary practices are poor. Thus, SLE in humans is typically a disease of members of the lower socio-economic groups (Monath and Tsai, 1987). *C. nigripalpus* is the epidemic vector of SLE virus in Florida. *C. tarsalis*, the primary vector for WEE viruses, is also the epidemic vector for SLE virus in the western U.S. Thus, the transmission of SLE and WEE viruses may occur concurrently in the western U.S. It is also for that reason that many of the ecological and environmental conditions that favor transmission of SLE virus are similar to those conditions that favor the transmission of WEE viruses (i.e., irrigated or flooded dryland areas).

The ambient temperature and rainfall profoundly influence the activity of SLE virus (Monath and Tsai, 1987). The requirement of a high ambient temperature is consistent with the epidemic curves of SLE outbreaks that occurred in central Florida in 1977 and in Houston, Texas in 1980. These epidemic curves show that incident cases occurred during mid-summer through late fall, specifically appearing first in July and continuing through August and September. High ambient temperatures early in the year appear to favor transmission of SLE virus by decreasing the amount of time required for the gonotrophic cycle, larval maturation, and the extrinsic incubation period of SLE virus. In laboratory studies, SLE virus was transmitted more efficiently by *C. tarsalis* and *C. pipiens* when the vectors were incubated at high ambient temperatures (Hardy, 1986). *C. quinquefasciatus* is not an efficient transmitter of SLE virus normally; however, high ambient temperatures in southern California may have increased the vector potential of *C. quinquefasciatus* during an epidemic of SLE in Los Angeles in 1984.

For more information about the epidemiology and ecology of SLE, you are referred to the supplemental readings listed in **Appendix 1**.

Bacterial Diseases

Ecology of Anthrax

Anthrax is an infectious disease caused by *Bacillus anthracis*. Historically, the geographic distribution of anthrax has been global. Now, the incidence is lower in much of the western hemisphere, but it is still a significant problem in parts of Africa and eastern Europe. Many domestic and wild animals, as well as humans, are susceptible to anthrax (Hugh-Jones, 1999). Ruminants and pigs are the most commonly affected food production animals, but horses located in the same environment also may acquire the infection (Mosier and Chengappa, 1999).

"For the control of [anthrax] epidemics, it is important to understand not only the pathogenesis and interactions of *B. anthracis* with host animals but also the **ecology of the spores**." (Dragon and Rennie, 1995). Generally speaking, anthrax epidemics occur during the summer months in which there are dry periods that are punctuated by prolonged periods of intense rain. The "incubator area" hypothesis, one attempt to explain why anthrax outbreaks occur, is based on observations that alkaline soil pH, high soil moisture, high concentrations of organic matter in the environment, and ambient temperature in excess of 15.5ºC provide a microenvironment that promotes cycling of *B. anthracis* spores, the end result of which is an increase in the exposure of susceptible hosts to infective doses of spores and more outbreaks of anthrax (Van Ness, 1971). The validity of this hypothesis continues to be challenged.

Prolonged rainfall obviously promotes runoff and pooling of standing water. A **proposed role** of water in anthrax epidemics is the collection (aggregation) and concentration of spores in spore "storage areas", a term that is intended to describe the stage of the life cycle of *B. anthracis* more accurately than does the term "incubator areas" (Dragon and Rennie, 1995). The surface of the *B. anthracis* spore is highly hydrophobic. Thus, the spores are resistant to dissolution by water and may be tranported in clumps of organic matter by runoff to standing pools of water. Subsequently, dry weather leads to evaporation of the standing pools of water and concentration of floating anthrax spores as these pools of water gradually shrink. The high buoyant density of *B. anthracis* spores provides an opportunity for the spores to adhere to vegetation as the vegetation resurfaces during evaporation of the surrounding water. Dragon and Rennie (1995) summarized the effects of water on anthrax spores essentially in three steps: (1) successive cycles of run-off and evaporation concentrate anthrax spores in storage areas, (2) evaporation redistributes the spores from the soil onto vegetation, and (3) susceptible herbivores consume the contaminated vegetation, become exposed to *B. anthracis* , and some develop anthrax.

Several ecological observations have been made during outbreaks of anthrax (Fox et al., 1973; Fox et al., 1977; Gainer and Saunders, 1989; Lindeque and Turnbull, 1994; Dragon and Rennie, 1995; Turner et al., 1999). The consistency of these observations varies, not only in reference to outbreaks that have occurred in livestock, but also in reference to outbreaks that have occurred in wildlife species. Cycles of rainfall and drought, specific grazing conditions, and specific soil types have been observed, but have **not** been observed consistently during outbreaks of anthrax.

For more information about the epidemiology and ecology of anthrax, you are referred to the supplemental readings listed in **Appendix 1**.

Ecology of Leptospirosis

Leptospirosis is a globally-distributed, bacterial disease of domestic and wildlife species as well as humans. The parasitic strains of these spirochaetes are found in animal species, and the saprophytic strains are found in water (Ellis, 1986; Ellis and McDowell, 1994). For taxonomic and epidemiological purposes, the parasitic leptospires are further divided into 23 serogroups and 212 serovars. Leptospiral infections also are broadly categorized into host-adapted and non-host adapted infections (Heath and Johnson, 1994). Host-adapted infections are endemic and are characterized by high seroprevalance of subclinical infections, primarily in intensively managed livestock production systems. Non-host-adapted infections are sporadic, of low seroprevalence, and the economic impact of non-host-adapted infections is lower than that of host-adapted infections (Ellis, 1994; Ellis and McDowell, 1994).

Environmental conditions are important in the survival of leptospires outside the host (Ellis and McDowell, 1994; Ellis, 1997). Prolonged viability of leptospires in the environment increases the risk of contact between leptospires and susceptible hosts, assuming that the host factors and pathogen factors involved in the transmission of leptospires remain equal. The optimum environmental conditions for survival of leptospires outside the host are warmth, moisture, and a nearly neutral pH (Michna, 1970; Ellis, 1986; Zaitsev et al., 1989; Ellis, 1994). Leptospires can survive for 30 days in sterile tap water and for 14 days in moist environments such as soils, mud, swamps and other watercourses that are relatively static. Water serves two functions in the transmission of leptospirosis. Water aids in the survival and dissemination of leptospires, as well as the actual process of a susceptible host becoming infected. Moving waters provide the force that promotes further physical spreading of leptospires within a contaminated environment, as well as dispersal from a contaminated environment to a non-contaminated environment (Ellis, 1997). Normally, intact skin provides an effective barrier against invasion by leptospires, but after the skin has become softened due to prolonged immersion in water, leptospires can penetrate the skin more easily and establish infection within the host.

A number of investigations of the survival of leptopires under various environmental conditions have been done. pH values outside the range of 6.0 to 8.4 are clearly unfavorable to survival of leptospires. On the other hand, the effect of soil pH values within the aforementioned range on the duration of survival of leptospires has not been shown to be consistent. This inconsistency is due possibly in part to differences in the methods that have been used to investigate the relationship between pH and the duration of survival of leptospires.

Regarding temperature and other physical agents, the ambient temperature, the body temperature within an infected host, and the temperature at which tissue specimens from an infected host are held all affect the viability of leptospires. Most of the investigations of the affect of temperature and other physical agents on survival of leptospires have been done in the laboratory. While it is not evident that as many similar investigations in natural environments have been undertaken, it is assumed that similar ambient temperatures (versus intra-uterine temperatures, e.g.) would have the same affect on the survival of leptospires in the environment as in the laboratory. In the laboratory, leptospires are susceptible to low and high temperature extremes, specifically temperatures less than minus 8.0ºC and greater than 50.0ºC. Leptospires survive for several hours at temperatures that are less than minus 18ºC and greater than 40ºC. In diagnostic specimens, leptospires die rapidly in tissues and body fluids, unless the tissues are maintained at plus 4.0ºC. Leptospires survive in tissues at 40ºC, but they die in tissues that are held at 30 to 39ºC (Rocha, 1998). This latter temperature range is the same range to which dead fetuses are exposed prior to being expelled by their dams; thus, the range may be one of the difficulties in isolating leptospires from carcasses during laboratory diagnosis of leptospirosis (Ellis, 1992). Leptospires are highly susceptible to dessication, UV light, and UV sunlight. They survive for 3 to 10 hours in a dry environment, for less than 0.2 hour when exposed to UV light, and less than 1.0 hours when exposed to UV sunlight (Michna, 1970; Faine, 1994).

Thus, the factors that determine the survival of some serovars of leptospires in the environment are the pH, ambient temperature, moisture, constituents of the soil, and the presence of other naturally-occuring, microbial competitors (Okazaki and Ringen, 1957). Regardless of the duration of survival of leptospires under specific environmental conditions, a key issue is whether they continue to remain infective to susceptible hosts under these conditions. Lastly, environments that favor either the maintenance, or the proliferation, of rodent and rat populations also favor the increased environmental contamination by leptospires. Examples of these environments are sugar cane farms specifically, farms in general, grain stores, and sewage plants (Ellis, 1997).

For more information about the epidemiology and ecology of leptospirosis, you are referred to the supplemental readings listed in **Appendix 1**.

Parasitic Diseases

Ecology of Cryptosporidiosis

Transmission of *Cryptosporidium parvum* is a complex maze that involves several reservoirs of infection. For purposes of this paper, transmission of *C. parvum* can be divided roughly into two types, waterborne transmission and non-waterborne transmission.

Cryptosporidiosis has been recognized with increasing frequency as a waterborne infection during the past 25 years (Rose et al., 1997).

Two sources of water involved in waterborne transmission of cryptosporidiosis are drinking water and recreational water. *C. parvum* spores are present in 65 to 97 percent of surface waters including rivers and lakes throughout the US (LeChevallier, 1991). Water that was collected from rivers, lakes, springs and beneath the ground has been implicated as the source of drinking water in at least five, well-documented outbreaks of water-borne cryptosporidiosis in the US (Rose et al., 1997). Because of the small size and density of *Cryptosporidium spp.* oocysts, approximately one year may pass before the suspended oocysts settle to the bottom of a 20 meter deep reservoir that contains standing water (versus flowing water). Thus, any anticipated reduction in the number of oocysts in surface water through natural sedimentation is not practical. However, properly operated, conventional water treatment methods can remove 99 percent of the oocysts, and microfiltration and ultrafiltration membrane processes may remove 100 percent of the oocysts.

Regarding the rivers, lakes, springs, and ground water that were implicated as the sources of the drinking water in the five outbreaks of water-borne cryptosporidiosis in the US, all of these sources were considered to be pristine sources prior to the outbreaks, indicating that only chlorination of the water was necessary to satisfy the regulatory requirements. The contaminated water was linked eventually to suboptimal treatment of water that was intended for human consumption. It was determined later that the water that was treated <u>did</u> meet some of the Environmental Protection Agency's requirements that were in effect at the time of the outbreaks. Wastewater was the suspected contaminant in two of these five outbreaks; however, the sources of contamination in the remaining three outbreaks remain unknown.

The Milwaukee, Wisconsin outbreak of cryptosporidiosis in 1993 was the largest reported cryptosporidiosis outbreak in US history (MacKenzie et al., 1994). Two rivers that discharge into Lake Michigan, the source of drinking water in this outbreak, became swollen after heavy rain. The rivers drain into the area of Lake Michigan where the water treatment facility collected its untreated water that was intended to undergo purification subsequently. The watersheds of the two rivers were sites for human sewage discharges, abattoirs, cattle grazing ranges, and environments with numerous species of wild and domesticated animals. However, neither the source nor time of entrance of oocysts into the water treatment facility was ever determined. Seven possible water-treatment deficiencies were described for this Milwaukee outbreak, as well as other outbreaks (Rose et al., 1997). Only <u>one</u> of the seven deficiencies in the Milwaukee outbreak and two other outbreaks was related to a naturally occurring event (i.e., heavy rain; melting snow). In regards to natural events, it was suggested that successful forecasting of events that may favor outbreaks of cryptosporidiosis (e.g. floods) would be beneficial to personnel who are involved in water treatment activities.

In addition to outbreaks of cryptosporidiosis in which the source was contaminated drinking water, there are many reported outbreaks of cryptosporidiosis due to contaminated recreational waters in the US and abroad. Recreational waterborne outbreaks have been linked to public swimming pools. Children were identified as the primary victims in the majority of these outbreaks. The source of cryptosporidium in recreational waterborne outbreaks were mostly fecal accidents in combination with inoperable swimming pool filters and, in one case, overflow of a **human** sewage system into the swimming pool. In general, these recreational water-borne outbreaks were not associated with the source of the water itself. Thus, the outbreaks were limited in their geographic distribution. As was the situation with outbreaks due to contaminated drinking water, disinfection of the swimming pools failed to inactivate the oocysts. Instead, the outbreaks were suppressed by closing and draining the contaminated pools, and the filters in the pools either were cleaned or replaced (Rose et al., 1997).

For more information about the epidemiology and ecology of cryptosporidiosis, you are referred to the supplemental readings listed in **Appendix 1**.

Discussion

Infectious diseases that are discussed frequently as potential animal health problems during natural disasters are mosquito-borne diseases, anthrax, leptospirosis, cryptosporidiosis, giardiasis, botulism, and hoof rot (Heath, 1999). A critical issue is whether the risk of these diseases during natural disasters truly increases, or whether the risks are more perceived than they are real. Plausible explanations of potential risks lie in part in the complex ecology and epidemiology of these diseases. If the risk of these diseases is remote, an awareness of the remote risks will permit those involved in the management of animals in natural disasters to direct their resources towards issues that truly are of utmost importance.

There is no question that outbreaks, even major outbreaks, of these infectious diseases in animal populations do occur occasionally, but there are few, rigorously controlled, scientific investigations to evaluate the incidence of the diseases during natural disasters. Although such studies are not readily available, a fundamental understanding of the ecology of these diseases will provide assistance in placing the risk of occurrence of these diseases in proper perspective during natural disasters. Many of the published investigations of outbreaks of these infectious diseases reported a variety of ecological and climatic conditions that were observed during the outbreaks **(Table 3)**. For example, some of the weather conditions that have been observed during outbreaks of arbovirus disease are increased precipitation, increased ambient temperature, and high relative humidity. However, these same conditions also may be observed when there are no outbreaks of disease. Regardless of the amount of precipitation, arbovirus diseases generally are restricted to specific months of the year, depending on the specific disease and the geographic region. The occurrence of anthrax outbreaks may be as much, if not more, a function of specific characteristics of the soil rather than changes in specific climatic conditions. The pH appears to have received the greatest amount of attention in the investigation of environmental factors that influence leptospirosis. The influence of climatic conditions on waterborne outbreaks of cryptosporidiosis appears to be even more vague than the influence of climatic conditions on the arbovirus diseases, anthrax, and leptospirosis.

Floods and hurricanes are frequently followed by a proliferation of mosquitoes and by concomitant requests from residents and government agencies to apply insecticides. However, many of the species of mosquitoes that proliferate during these disasters are nuisance mosquitoes, rather than mosquitoes that are competent vectors of infectious diseases (Centers for Disease Control and Prevention, 1994; Nasci and Moore, 1998). Nuisance mosquito species are **not** a significant threat to animal health or human health (Centers for Disease Control and Prevention, 1993a). Regardless, it has been suggested that a provision to monitor the following two parameters should be included in disaster response policies for animals and humans: **(1)** monitor increases in prevalence of mosquito species that can transmit infectious agents and **(2)** monitor the risk of arboviral diseases in those geographic regions that are affected by natural disasters (Nasci and Moore, 1998).

Table 3. Ecological and climatic **observations**[1] during: (1) enzootic and epizootic transmission of selected arbovirus diseases that are prevalent in the U.S., Canada, Mexico, and Central America, (2) epizootic transmission of anthrax in livestock and wildlife in the U.S., Canada, and Africa, and (3) epizootic transmission of leptospirosis and cryptosporidiosis.

	Viral (i.e., Arbovirus)				**Bacterial**			**Parasitic**
	EEE	**WEE**	**VEE**	**SLE**	**Anthrax**		**Leptospirosis**	**Cryptosporidiosis**
Ecological Observation					**Livestock**	**Wildlife**		
Increased annual precipitation	+		+					
Increased annual ambient temperature		+	+	+				
High relative humidity (90%)		+	+		+			
Incomplete drainage-lake type hydrographic region	+							
Wooded land on the affected premises	+							
Sunlit grassy marshes, open pools of stream beds, especially in irrigated areas		+		+				
Permanent bodies of water; lowland swamps; coastlines			+					
Mid-summer to late-summer season	+	+	+		+	+		
Late summer and fall season				+				
High ambient temperature immediately preceding and during the outbreak					+	+		
A “dry period” that was followed immediately by a “wet period”					+			
A “dry period” that was <u>not</u> followed by a “wet period”					+			
A wet spring that was followed by a summer drought						+		
Poor grazing conditions					+	+		
Optimum grazing conditions						+		
Alluvial soils derived from loam and clay sediments (alkaline pH)					+			
Calcareous soils					+			
Soils with poor drainage of surface water					+			
Disruption of soils during construction projects in which earth was moved					+			

Table 3. Ecological and climatic **observations**[1] during: (1) enzootic and epizootic transmission of selected arbovirus diseases that are prevalent in the U.S., Canada, Mexico, and Central America, (2) epizootic transmission of anthrax in livestock and wildlife in the U.S., Canada, and Africa, and (3) epizootic transmission of leptospirosis and cryptosporidiosis.

	Viral (i.e., Arbovirus)				**Bacterial**			**Parasitic**
	EEE	**WEE**	**VEE**	**SLE**	**Anthrax**		**Leptospirosis**	**Cryptosporidiosis**
Ecological Observation					**Livestock**	**Wildlife**		
Poorly drained swamps that were located directly on, or immediately adjacent to, affected premises					+			
Nearly neutral pH in the environment							+	
Moist environment containing static water							+	
Warm ambient temperature							+	
Absence of UV light, UV sunlight							+	
Floods (e.g., excessive short-term rainfall)								**?**

[1] Use of the word " **observation(s)** " should not be equated with use of the words "risk factor(s)". EEE, eastern equine encephalitis; WEE, western equine encephalitis; VEE, Venzuelean equine encephalitis; SLE, St. Louis encephalitis. **?**, inconclusive

The three arbovirus diseases of primary concern to human health in the U.S. are WEE, EEE, and SLE. Both WEE and EEE also are of concern to the health of horses, other livestock, and some domesticated avian species. Each of these diseases has a distinct ecology that involves different mosquito species, different amplifier hosts, and other factors that affect (by either promoting or inhibiting) transmission of the infection. While concerns about the risk of disease transmission by vector species may increase during natural disasters, natural disasters in the continental United States have been accompanied only rarely by outbreaks of mosquito-transmitted diseases of humans (Centers for Disease Control and Prevention, 1993a; Centers for Disease Control and Prevention, 1994).

One investigation of the incidence of vector-borne diseases of animals and humans was done during 12 natural disasters that occurred in the United States from 1975 to 1997 (Nasci and Moore, 1998). The three types of natural disasters that were examined were floods, hurricanes, and tropical storms. Surveillance for arboviruses was undertaken during 11 of the 12 natural disasters **(Table 4)**. Although most of the disasters, and therefore the surveillance programs, were confined to three states or fewer, as many as seven states were involved in some of the 11 disasters. Arbovirus activity (i.e., for WEE, EEE, SLE) was detected in mosquitoes and sentinel chickens during nine of the 11 disasters in which surveillance was undertaken. What is not clear is whether a similar level of arbovirus activity also was detected in these same nine locations during years in which there were no natural disasters. Regardless, human cases of arbovirus disease were diagnosed during only two of the nine natural disasters during which arbovirus activity was detected. There were 55 reported cases of WEE and 12 reported cases of SLE in humans in North Dakota and Minnesota during the Red River flood of 1975, and there was only one case of EEE in humans during Hurricane Fran in North Carolina in 1996 **(Table 4)**. Surveillance for SLE and dengue fever in humans during Hurricane Andrew in 1992 failed to show an increased incidence of either disease (Heath, 1999). During Hurricane Hugo in 1989, the most prevalent problem related to insects was not infectious diseases, but insect bites and stings from **nuisance** mosquitoes, hornets, wasps, and bees.

Regarding the veterinary cases that were examined by Nasci and Moore, arbovirus diseases were diagnosed during four of the nine disasters in which arbovirus activity was detected in mosquitoes and sentinel chickens. WEE was diagnosed in approximately 281 animals during the Red River flood of 1975. The exact number of animals that were diagnosed with arbovirus disease during the other three natural disasters was not reported **(Table 4)**. While the number of disease outbreaks in animals was greater than in humans (i.e., four versus one), there was only one natural disaster involving flooding to which significant animal morbidity and mortality due to arbovirus disease was attributed (i.e., WEE in North Dakota and Minnesota during the Red River flood of 1975). Each of the other three natural disasters lead to excessive accumulations of water, but in addition to excessive accumulations of water, outbreaks of arbovirus disease can be influenced by other factors such as topography, the types of bodies of water, and the season of year. In comparison to the Red River flood, the differences in these factors during the other three natural disasters may account for the lower number of incident cases of vector-borne disease that were identified.

Table 4. Veterinary and human cases of vector-borne diseases during 12 natural disasters that lead to excessive accumulations of water in the continental United States from 1975 through 1997.*

Year	Region	Event	Surveillance?	Pathogen or Disease	Human Cases	Veterinary Cases
1975	ND, MN	Red River flood	Yes	WEE✞ in mosquitoes	55 WEE; 12 SLE⌖	281 WEE (estimated)
1989	Southeastern U.S.	Hurricane Hugo	Yes	EEE✠ in mosquitoes	None	No data
1992	FL, LA	Hurricane Andrew	Yes	None	None	None
1993	AZ	Gila River flood	Yes	SLE, WEE in mosquitoes	None	None
1993	Midwestern U.S.	Mississippi, Missouri River flood	Yes (7 states)	WEE in SD; SLE in IL	None	None
1994	AL, FL, GA	Tropical storm Alberto	Yes	EEE in AL and FL	None	EEE in horses and emus
1995	CA	Winter and spring floods	Yes	WEE, SLE in sentinels	None	WEE
1996	CA	Winter flood	Yes	WEE, SLE in chickens; WEE in mosquitoes	None	None
1996	OR, WA	Winter flood	No	No	None	None
1996	NC	Hurricane Fran	Yes	EEE in mosquitoes	1 EEE	EEE in horses
1997	CO	Summer floods	Yes	WEE in chickens	None	None

Table 4. Veterinary and human cases of vector-borne diseases during 12 natural disasters that lead to excessive accumulations of water in the continental United States from 1975 through 1997.*

Year	Region	Event	Surveillance?	Pathogen or Disease	Human Cases	Veterinary Cases
1997	ND, MN	Red River flood	Yes (sporadic)	None reported	None	None

Source: From Nasci and Moore, *Emerging Infectious Diseases*, 1998. Reproduced with permission. *Surveillance data collected by the Division of Vector-Borne Infectious Diseases, Centers for Disease Control and Prevention (CDC). State and local health departments assisted during emergency response. Federal Emergency Management Agency and Emergency Response Coordination Group, National Centers for Environmental Health, CDC, provided field support. ✞Western equine encephalitis. ✟St. Louis encephalitis. ✠Eastern equine encephalitis.

Another investigation of the incidence of vector-borne diseases of horses and humans during a natural disaster was done in Nebraska in 1994-1995, following the Midwest flood of 1993 (Janousek and Kramer, 1998). WEE virus was isolated from 1.29 percent of 2,788 pools of Culex mosquitoes, and SLE virus was isolated from 0.14 percent of 1,359 pools. All arboviruses that were isolated were from *Culex tarsalis* Coquillett, a major vector of WEE and SLE. No incident cases of WEE were reported in horses in Nebraska in 1994-1995. Neither were there reported cases of WEE or SLE in humans in Nebraska in 1994-1995. In yet another investigation, there was no reported EEE and SLE virus activity during Hurricane Andrew in 1992. This surveillance program involved monitoring wild birds for exposure to virus (Centers for Disease Control and Prevention, 1993a).

Hurricane Floyd in North Carolina in 1999 is one of the most recent natural disasters that involved widespread flooding of animal and human populations. The median number of cases per year of EEE, WEE, and SLE in humans in North Carolina from 1964-1997 was 1.5, 0, and 1.0, respectively (Centers for Disease Control and Prevention, 1999). During the year of Hurricane Floyd and the two years after Hurricane Floyd (i.e., 2000, 2001), there was no evidence that the number of human cases of EEE in North Carolina increased (North Carolina Department of Environment and Natural Resources, 2001). The incidence of WEE and SLE in North Carolina was rare prior to Hurricane Floyd and remained so after the hurricane. Because these data for years 1999, 2000 and 2001 do not indicate an apparent increase in the number of cases of arbovirus disease in humans in North Carolina, the incidence of these diseases could not be linked to Hurricane Floyd in a temporal sense.

In general, arbovirus surveillance in animal populations in the U.S appears to be much less refined than it is in the human population (Personal communication, Eileen Ostlund, USDA National Veterinary Services Laboratory, 2001). Thus, a comparison of animal surveillance data during the pre-natural disaster, natural disaster, and post-natural disaster periods would be significantly less reliable than a comparison of human surveillance data from these same periods of time. Nonetheless, it seems appropriate to conclude that during the past 25 years there was only one natural disaster involving flooding to which significant **human** and **animal** morbidity and mortality due to arbovirus disease was attributed. That disaster was the Red River flood of 1975.

The weather conditions that have been observed most frequently during anthrax outbreaks differ depending on whether the affected animals are domestic species or wildlife. During anthrax outbreaks in livestock, high ambient temperatures, calcareous soils, and a dry period which may or may not have been followed by a wet period are some of the conditions that have been observed most frequently **(Table 3)**. During anthrax outbreaks in wildlife, a wet spring followed by a summer drought, and either poor grazing conditions or optimum grazing conditions have been observed frequently. Weather conditions that are common to anthrax outbreaks in both livestock and wildlife are mid-summer to late summer season, high ambient temperature immediately preceding and during the outbreak, and poor grazing conditions. The extent to which any one or various combinations of these factors increases the risk of outbreaks of anthrax is not clear. It seems appropriate to conclude that natural disasters, particularly floods and droughts, would not favor outbreaks of anthrax since neither floods nor droughts produce the wet/dry cycle which has been hypothesized to be essential for anthrax outbreaks to occur.

Cryptosporidiosis is endemic in animals in the U.S. Localized outbreaks are common, but scientific reports of widespread outbreaks of cryptosporidiosis in animals are rare. The largest outbreaks of cryptosporidiosis in humans in the U.S. in which water was the source of the infection have **not** been linked to contamination of the water by waste products from domestic animal populations, neither during natural disasters such as flooding, nor otherwise. The "suspected cause" of each outbreak was categorized either as : **(1)** a water treatment deficiency (ies), **(2)** contamination by human sewage, or **(3)** undetermined. Regardless of the source of each outbreak, the solution to reducing waterborne outbreaks of cryptosporidiosis in humans lies in adhering to firmly established, conventional water treatment practices (Rose et al., 1993; Rose et al., 1997). There are other potential routes of transmission of cryptosporidiosis from animals to humans, but these routes of transmission are influenced minimally to none by natural disasters such as flooding.

There is little scientific evidence that links natural disasters in the U.S. to significant and consistent increases in the incidence of infectious diseases of animals that are frequently discussed during these disasters (e.g., vector-borne diseases, anthrax, leptospirosis, cryptosporidiosis, etc.). The massive destruction of the crop ecosystems due to Hurricane Floyd in North Carolina in 1999 suggests that neither human-derived ecosystems nor natural ecosystems can be protected with ease from the path of destruction of natural disasters such as floods, hurricanes, extreme heat, extreme cold, and fires. Thus, the destruction caused by the natural disasters might in fact destroy the human-derived and natural ecosystems which harbor infectious disease pathogens and their vectors. Consequently, the risk of outbreaks of infectious disease in geographic regions that have been overcome by natural disasters may not increase. Instead, the risk of outbreaks of infectious disease in such regions may decrease, either temporarily and sometimes permanently.

Non-Infectious Hazards of Concern During Natural Disasters

Introduction

Two *non-infectious* diseases of animals that are commonly discussed during natural disasters are aspiration pneumonia and injuries (Heath, 1999). Morbidity and mortality due to non-infectious diseases can be attributed to excesses of water, excesses of wind, lightning, deficiencies of water, heat extremes, fires and earthquakes. The animal health problems that may arise due to these natural disasters include drowning, hyperthermia, burns, hypothermia, and traumatic injuries **(Figure 1)**.

Water Excesses (Floods)

Several thousand dairy cows perished during floods in Snohomish Valley, Washington in 1991 (Heath, 1999). Approximately 1,200 dairy cows perished during floods in Tilamook, Oregon in 1996. During 1997, approximately 90,000 beef cows died during floods in Minnesota, North Dakota, and South Dakota.

It is likely that some of the most recent and significant economic losses from livestock deaths due to a natural disaster occurred during Hurricane Floyd in North Carolina in 1999. Approximately 40 of the 100 counties in North Carolina experienced flooding during Hurricane Floyd (Hudson et al., 2001). The total estimated value of livestock that died was $13 million **(Table 5)**. However, the total number of livestock deaths ". . . represented a small percentage . . . " when compared to North Carolina's total livestock population (North Carolina Department of Agriculture and Consumer Services, 1999).

Morbidity and mortality of pets during floods appears to be much more rare than livestock deaths (Heath, 1999). Hypothermia is the most frequently reported illness of all animals that are rescued from floodwaters. The actual number of reports of pets that have drowned during floods is no more than ten (Heath, 1999). While this number would appear to be surprisingly small, it does seem to be consistent with there being minimal reference to companion animal mortality that may have been caused by Hurricane Floyd (Hudson et al., 2001). During Hurricane Floyd, a disaster relief management program was established by the North Carolina State University College of Veterinary Medicine for approximately 467 animals (375 dogs, 75 cats, and 17 animals of other species) that were affected by the flood. The source of at least 130 of the animals that were managed by the disaster relief management team was a crowded county animal shelter that became flooded. That the source of these animals was a crowded shelter suggests that many of the animals may have been separated involuntarily from their owners <u>prior to</u> the onset of Hurricane Floyd. Still other animals may have become separated from their owners **voluntarily** (i.e. abandoned) <u>prior to</u> the onset of Hurricane Floyd.

Table 5. Estimated value of livestock and aquaculture species that died during Hurricane Floyd in North Carolina in 1999.

Livestock	Number of Deaths	Cost ($)
Hogs	28,000	$1,680,000
Chickens	2,107,857	$3,583,357
Turkeys	752,970	$7,153,215
Cattle	1,180	$495,600
Aquaculture	NA	$100,000
Total		$13,012,172

Source: http://www.agr.state/nc.us/state/c%26lloss.htm . NA = not available.

Table 6. Anatomical location of injuries on horses admitted to an emergency health care facility after Hurricane Andrew in Florida in 1992.

Anatomical Location	Number of Injuries	Percent of Total
Lower hind limb	32	30.8
Torso	26	25.0
Upper hind limb	16	15.4
Head and neck	16	15.4
Upper fore limb	7	6.7
Eyes	7	6.7
Total	122	100.0

Source: Heath, *Animal Management in Disasters*, 1999. Revised, with permission.

Approximately 300 of the 450-plus animals that were admitted to the field hospital were adopted after Hurricane Floyd. Prior to adoption, 99 percent of the 300 animals had not undergone spay/neuter, and more than 99 percent had not been vaccinated against rabies. A logical question is whether the demand for the many types of fundamental health care services that were provided to the animals by this veterinary medical assistance team could be attributed more directly to animal abandonment and neglect that was present prior to Hurricane Floyd, rather than to problems that were caused directly by Hurricane Floyd? Technically speaking, it appears that many of the animals may have been "owned", but they were permitted to roam freely and received minimal veterinary medical care even prior to Hurricane Floyd (Personal communication, Lola C. Hudson, North Carolina State University College of Veterinary Medicine, 2001).

Morbidity in humans was investigated during a severe thunderstorm in central Texas during October, 1998 (Centers for Disease Control and Prevention, 2000). Of the 31 human deaths, 29 were attributed directly to the thunderstorm, and two deaths were attributed indirectly to the thunderstorm. Of the 29 deaths attributed directly to the thunderstorm, 83 percent (N = 24) were due to drowning, 10 percent (N = 3) were due to multiple traumatic injuries, 3 percent (N = 1) were due to hypothermia, and 3 percent (N = 1) were due to cardiac arrhythmia.

Wind Excesses (Hurricanes, Tornadoes)

Damage from hurricanes may be due to: (1) direct damage from winds and rains that generate flying debris and destroys structures, (2) direct damage from storm surge that leads to flooding, and (3) secondary damage from electrocutions and hazardous materials during activities that are aimed at restoration (Heath, 1999). Veterinary medical facilities are not immune to hurricanes and tornadoes. An intact veterinary infrastructure is an essential prerequisite for veterinary practices to provide services to injured animals and to coordinate relief efforts. Thus, the single most important veterinary issue after a hurricane is to reestablish compromised veterinary infrastructure.

The most prevalent health problem of animals that is caused by the wind forces generated during hurricanes is trauma. Traumatic injuries were common after Hurricane Andrew in Florida in 1992 and after Hurricane Fran in North Carolina in 1996. These injuries may be caused by flying objects during hurricanes, or they may result from direct encounters with scattered debris after the hurricane. Approximately 2000 pets received medical care for lacerations, fractures, and urinary tract infections after Hurricane Andrew. Lower-limb injuries were the most prevalent type of injury in horses **(Table 6)**. However, the baseline incidence (i.e., pre-hurricane) and the recovery-period incidence (i.e., post-hurricane) of these problems in the companion animals and horses should be considered in evaluating the proportion of these problems that should be attributed to hurricanes.

Both mortality and morbidity in humans were investigated during Hurricane Andrew in Louisiana during August 1992 (Centers for Disease Control and Prevention, 1993b). Of 17 human deaths attributed to Hurricane Andrew, only nine occurred after landfall; 67 percent of these were due to drowning, 11 percent were due to impact injury, 11 percent were due to crush injury, and 11 percent were not categorized. The most prevalent, non-fatal, health problem was categorized as cut/laceration/puncture wound, followed by strain/sprain. Sixty-percent of the non-fatal health problems were confined to the extremities.

Lightning

Livestock and horses are the domestic animals that are most likely to be struck by lightning because they reside outdoors (Heath, 1999). These animals are especially vulnerable if they are confined near large, free-standing trees. If the animals have free access to the base of the trees, they will congregate there during thunderstorms to protect themselves from rain and hail. If lightning strikes the trees, the electricity can be conducted through the animals' bodies enroute to the earth. Lightning stroke in cattle is frequently fatal.

Water Deficiencies (Droughts)

A practical definition of drought is when precipitation and other water resources fall below expectations, and this expectation is not met by a concomitant decrease in demand for water (Heath, 1999). Droughts may be the most expensive natural disaster to the animal industries in the U.S. because droughts tend to be more widespread in their geographical distribution (Heath, 1999). Decreased availability of forage, decreased growth rates, and decreased productivity are consequences of drought. During droughts, the greatest losses in cattle production are from starvation. However, the scarcity of forage during droughts means that livestock, horses, and wildlife may consume toxic plants. Drought-related poisonings of cattle include salt poisoning, urea poisoning, nitrate poisoning, cyanide poisoning, selenium poisoning, and rumen impaction (Heath, 1999; Howard and Smith, 1999).

Heat Extremes

Extreme heat is defined as an ambient temperature that is at least 5.5º C (i.e., 10º F) higher than the average high temperature for a specific geographical location (Heath, 1999). There are differences in the average temperature in different geographical locations and at different times of the year in the U.S; thus, the absolute temperature that should be used to define extreme heat varies with the location. Although extreme heat occurs typically during the summer months, high ambient temperatures even during the cool months may have the same adverse biological affects on animals, if the animals have not had an opportunity to adapt to these temperatures.

Hyperthermia, a body temperature of 42.0º C or 107.0º F, is a primary outcome of extremely high ambient temperatures. Cattle mortality due to hyperthermia occurred in California in 1977. The mortality increased significantly in a dairy herd during four consecutive days of intense heat, and a total of 725 milking cows perished. Similarly, the mortality increased significantly in feedlot cattle in Iowa and Nebraska during a period of intense heat in July 1995. A total of 10,000 feedlot cattle perished, 3,750 within a single day (Bopp, 2001; Wren, 1997). The estimated losses to livestock producers in central Iowa was $28 million, and the estimated losses to poultry producers was $25 million. Another indicator of the impact of extreme heat is an increase in rendering activity. The number of dairy cow carcasses that were rendered each week during a heat wave in Wisconsin increased from 400 to 15,000 (Heath, 1999).

Ruminants do not sweat, so panting is the mechanism by which they avoid abnormal increases in body temperature during environmental heat extremes. However, the increased air exchange increases their susceptibility to the bovine respiratory disease complex. Experience has shown that there are three critical factors that determine if feedlot fatalities will occur either directly or indirectly from heat extremes (Bopp, 2001). These three factors are: **(1)** intensity of heat, **(2)** duration of the heat extreme, and **(3)** opportunities at night for cattle to recover from exposure to the heat extreme. Intensity is a function of the ambient temperature in combination with humidity. Rainfall immediately prior to onset of a heat wave has been associated with high percent mortality because the humidity will increase greatly. Approximately 72 consecutive hours of extreme heat also has been associated with increased percent mortality. However, if the ambient temperature decreases at night to 70^0 F or lower, feedlot cattle can recover from the effects of exposure to the heat extremes that may have taken place during daylight hours. The lower ambient temperature, although transient, will diminish the cumulative biological effects of the heat.

Fires

Fires are classified as to whether they occur in buildings, wildlands, or elsewhere (Heath, 1999). The greatest impact of wildland fires (i.e., forest fires and wildfires) is on wildlife and the environment, not domestic animals and humans. However, as urban dwellers, who will be accompanied by their domestic animals, continue to encroach on natural environments, the impact of forest fires and wild fires on the domestic animal population may increase.

The number of reported injuries to animals from fires is low, suggesting that injuries that require medical therapy are rare. Burn injuries of horses and cats are reported more frequently than of other species. Reports of burn injuries in horses are most frequently of extensive wounds to the skin. The sequellae to burn injuries in horses include pulmonary edema, oliguria, polyuria, infections, and anemia.

Earthquakes

The reports of animal injuries following earthquakes are mostly anecdotal (Heath, 1999). Regardless, the impact of earthquakes on animal health in the U.S. appears to be small. Among the medical problems associated with the Northridge earthquake in California were bruises and fractures due to blunt-force trauma, wing-tip injuries to birds that had been housed in cages that became dislodged, lacerations from broken glass, and prolonged hiding. While specific numbers of injured animals have not been reported, the numbers do appear to be rather small. Infectious diseases, especially tetanus, are a sequela to traumatic injuries (e.g., lacerations). Generally speaking, horses are more susceptible to tetanus than are most other domestic species. Two non-medical problems that were observed during the Northridge earthquake were animals that strayed from damaged confinements and the excessive number of animals that became available for adoption.

Conclusion

There is little scientific evidence that links natural disasters that have occurred in the U.S. to significant and consistent increases in the incidence of infectious diseases of animals. The transmission of infectious diseases at a level that is sufficient to alter the incidence from a sporadic level to an enzootic level, and from an enzootic level to an epizootic level can be a complex mixture of natural and human-made events. The complexity of these events may be even more true for vector-borne infectious diseases which, again, are one group of diseases for which there seems to be significant concern during natural disasters (Heath, 1999).

There are numerous ecological and climatic factors that have been observed during epizootics of vector-borne and non-vector-borne infectious diseases. Some factors have been observed rather consistently. Others have been observed far less consistently. Regardless, mere observation of these climatic conditions during an outbreak does not necessarily imply that the outbreak was caused by the climatic conditions. Nor have these observations, in and of themselves, proven thus far to be very valuable in predicting whether an outbreak of infectious disease will occur during a natural disaster. Attempts to rationalize the relationship between weather, vector biology, and arboviral recrudescence have been summarized by a vector-borne disease entomologist as "... the hope that weather forecasting and weather analysis might eliminate the element of surprise in arboviral epidemics. The sobering truth is that arboviral epidemiology is a complex, multifactorial process, and that coincidental events involving some or all variables are the true precipitating factors for recrudescence ... an aggressive approach is clearly required to convert the wealth of random speculation on the weather/recrudescence relationship into useful information." (Reiter, 1988).

Based on data available from studies of natural disasters, the apparent morbidity and mortality in animal populations that has been caused by *non-infectious* diseases, while involving an almost insignificant percentage of the total population in affected geographical regions, is much higher than the apparent morbidity and mortality that has been caused by *infectious* diseases. One explanation is that, generally speaking, the sequence of events from a cause (e.g., excesses of water) to an effect (e.g., drowning) for non-infectious diseases is more simple, direct, and recognizable than the complex sequence of events that lead to major outbreaks of infectious diseases (e.g., EEE). The epidemiological evidence suggests that the potential economic impact of non-infectious diseases arising from natural disasters may be greater than the economic impact of infectious diseases. While vigilant surveillance for outbreaks of infectious diseases during natural diseasters should not be discouraged, there should be appropriate resources allocated towards the resolution of issues related to the prevention, control and treatment of non-infectious diseases during natural disasters.

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Appendix

Table 7. Supplemental readings about the ecology and epidemiology of various infectious diseases of animals that are of concern during natural disasters. ⌖
Title
Ecology and Epidemiology of Eastern Equine Encephalomyelitis
Ecology and Epidemiology of Western Equine Encephalomyelitis
Ecology and Epidemiology of Venzuelan Equine Encephalomyelitis
Ecology and Epidemiology of St. Louis Encephalitis
Ecology and Epidemiology of Anthrax
Ecology and Epidemiology of Leptospirosis
Ecology and Epidemiology of Cryptosporidiosis
⌖These readings will be available at http//www.aphis.usda.gov/vs/ceah/cei by summer 2002.

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